

Case Report

Fatal *Chromobacterium violaceum* septicemia in a South Indian adult

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Abstract

Chromobacterium violaceum is a rare human pathogen that causes potentially fatal infections especially in the tropical regions. Limited awareness about this pathogen and inappropriate antibiotic therapy are some of the factors contributing to the high mortality rate. To date there have been only eight cases reported from India of which only one is an adult. To the best of our knowledge, we report here the first case of a 40-year-old man from South India with septicemic *C. violaceum* infection and septic arthritis.

Key words: *Chromobacterium violaceum*; septicemia; septic arthritis

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Introduction

Chromobacterium violaceum is a Gram-negative bacillus that is a common inhabitant of water and soil and can be isolated from natural aquatic habitats in the tropical regions. In spite of its ubiquitous distribution, human infection due to *C. violaceum* is extremely rare. *C. violaceum* was first identified in 1881; its pathogenic potential was first described by Woolley in 1905, when he isolated it from a fatal infection in a buffalo [1] and the first case in humans was reported from Malaysia in 1927 [2]. Septicemia, visceral abscesses, skin and soft tissue infections, urinary tract infections, and diarrhea are some of the common manifestations. The mortality rate associated with these infections is very high. There have been about 150 cases reported worldwide, especially from the tropics, including eight cases from India [3]. We report *C. violaceum* septicemia and septic arthritis in a 40-year-old man from South India who had a fatal outcome.

Case report

A 40-year-old male farmer from South India presented with a 10-day history of high-grade intermittent fever with chills associated with pain and swelling of the right knee joint. Seven days into his illness he was admitted to the local health-care facility. One day prior to admission he had also developed worsening breathlessness and was hence referred to our institution. There was no history of cough,

expectoration, dysuria, diarrhea, or headache. There was no history of trauma to the affected knee joint in the recent past. The patient had been diagnosed to have systemic lupus erythematosus in 1997 and had been on chloroquine and intermittent corticosteroids. In 1999 he was treated with a six-month course of anti-tuberculous therapy for pulmonary tuberculosis, diagnosed by positive sputum culture. He also underwent a cemented total hip arthroplasty on the right side in 2001 when he was diagnosed to have bilateral avascular necrosis of the femoral heads, probably secondary to long-term steroid use. He was neither diabetic nor an alcoholic, and he was negative on HIV testing.

He was a farmer by occupation and lived in a rural village in South India where he used to do rice farming. He gave no history of recent travel or leisure activities such as swimming.

On examination he appeared toxic, was febrile with a temperature of 103°F, tachycardic with a pulse rate of 120 beats/minute, tachypnoic with a respiratory rate of 44 breaths per minute, and had blood pressure of 100/60 mm of Hg. Examination of the right knee revealed a warm, tender and swollen joint with evidence of an effusion. The remainder of the general and systemic examinations was normal except for bilateral basal crackles on auscultation.

A clinical diagnosis of septic arthritis of the right knee joint with systemic inflammatory response syndrome (SIRS) was made. The etiological agents

usually implicated in septic arthritis were considered, including *Staphylococcus aureus* and *Streptococcal pneumoniae*. In view of the immunosuppression secondary to steroid use, Gram-negative organisms, particularly *Burkholderia pseudomallei*, were also considered. Other acute febrile illnesses endemic in the tropics such as malaria, dengue, leptospirosis and rickettsial infections were considered less likely as none of them cause septic arthritis.

On admission, his haemoglobin was 9.8 g/dL, total WBC count was 15,700 cells/ μ L with 87% neutrophils, 6% lymphocytes and 7% monocytes. Biochemical parameters revealed normal renal functions, elevated liver enzymes, elevated total and direct bilirubin, and reversed albumin/globulin ratio. Serological tests for Hepatitis B, C and HIV were negative.

Chest radiograph, electrocardiography (ECG), and sonography of the abdomen were essentially normal. Ultrasound screening of the right knee showed a moderate effusion with internal echoes suggestive of an infection. Right knee joint aspiration revealed turbid fluid with a cell count of 47,000 cells/ μ L and 98% polymorphs. Four blood cultures (collected on the first and second hospital days) and the synovial fluid aspirate from the right knee joint (obtained at admission) yielded the same organism, *C. violaceum*. Blood and synovial fluid specimens were inoculated on sheep blood agar and Mac Conkey agar plates. The sheep blood agar was incubated with 5% carbon dioxide at 37°C. The Mac Conkey agar was incubated

at 37°C aerobically. After 24 hours, fine, violet colonies were observed on both media (Figure 1). After 48 hours of incubation, 0.5 mm to 1 mm beta-hemolytic, violet-pigmented colonies were visible on sheep blood agar. The conventional biochemical test characteristics were consistent with the identification of *C. violaceum* [4].

Antibiotic susceptibility testing of the *C. violaceum* was performed by E-test as indicated by the manufacturer [5]. The isolate was susceptible to cotrimoxazole (MIC 0.19 μ g/ml), ciprofloxacin (MIC 0.012 μ g/ml), imipenem (MIC 1.0 μ g/ml), and meropenem (MIC 0.125 μ g/ml), and moderately susceptible to ceftazidime (MIC 24 μ g/ml). The patient was given empirically piperacillin/tazobactam on day one. However, his condition progressively deteriorated and he became hypotensive and required mechanical ventilation. On day two the antibiotic was changed to meropenem but there was no clinical improvement; the patient progressively went into multi-organ failure and succumbed to his illness on the third day. The cultures were confirmed on the fifth day.

We did not conduct any epidemiologic, clinical, laboratory or environmental investigation in the area from which the patient came.

Discussion

C. violaceum is a motile Gram-negative, non-spore forming, facultative anaerobic rod-shaped bacterium.

Figure 1. Fine violet-pigmented colonies observed on the nutrient agar plate after 24 hours of incubation, typical of *C. violaceum*

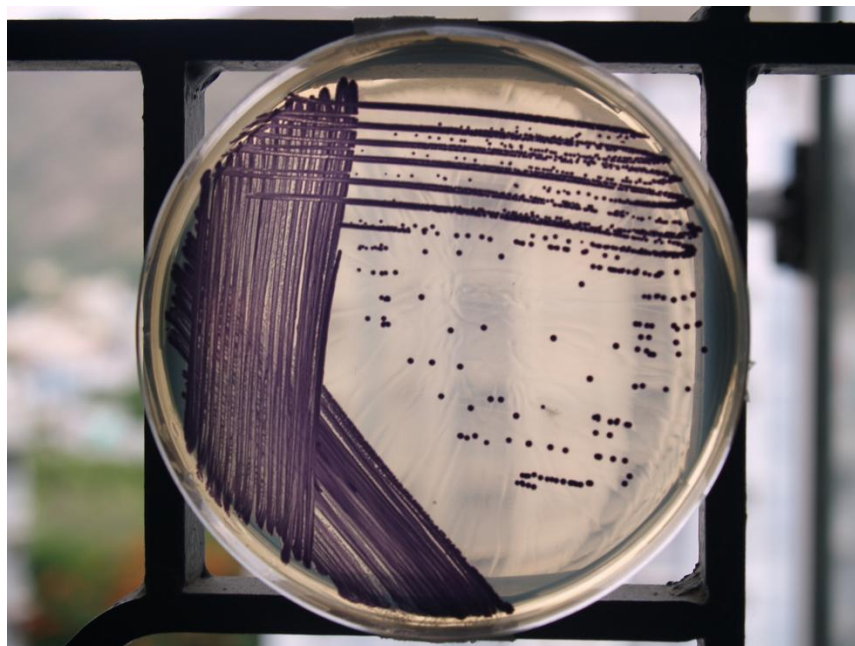


Table 1. Cases of *C. violaceum* infection reported from India

Case	Year	Geographic location	Age & Sex	Clinical presentation	Specimen	Outcome	Reference
1	1979	Andhra Pradesh	4 / M	Septicaemia Meningitis Ulcers	Blood, Skin lesions	Fatal	Annapurna <i>et al.</i> , 1979 [19]
2	1987	Karnataka	NB / M	Meningitis	CSF	Fatal	Shetty <i>et al.</i> , 1987 [13]
3	2000	Karnataka	2 yr 10 months / F	Diarrhoea	Stool	Recovered	Ballalet <i>et al.</i> , 2000 [20]
4	2002	Karnataka	2 months / F	Pustules Ear discharge Septicaemia Meningitis	Blood Liver pus Sinus discharge Urine	Fatal	Chattopadhyay <i>et al.</i> , 2002 [21]
5	2002	Karnataka	8 days	Pustules Septicaemia Multiple abscesses	Blood Skin Ear	Fatal	Shenoy <i>et al.</i> , 2002 [22]
6	2002	Chandigarh	6.5 yrs / M	Septicaemia Pustules	Blood Skin biopsy	Recovered	Ray <i>et al.</i> , 2004 [3]
7	2003	West Bengal	24 yrs / M	Abscess leg	Pus	Recovered	Dutta <i>et al.</i> , 2003 [23]
8	2008	Kerala	6 month / M	Septicaemia Skin pustules Broncho pneumonia	Blood Pus from skin lesions	Recovered	Vijayan <i>et al.</i> , 2009 [24]

It is the exclusive species of this genus that causes human disease. Most strains of the organism produce a pigmented violacein that gives the colonies a characteristic violet color and the organism its name. Rare non-pigmented strains also exist.

C. violaceum is ubiquitous and inhabits soil and stagnant water. Infection with this organism is largely confined to the tropical and subtropical regions. Since the discovery of the first case, there have been more than 150 cases reported worldwide. In addition to the Indian subcontinent, the majority of cases have been reported from southeast Asia, South America, Australia, and the southeastern United States, particularly Florida [6]. There have been eight cases

reported from India prior to this case [3] (Table 1). The route of entry of this organism still remains unclear. Most patients have a history of trauma or wounds which have been contaminated with soil or stagnant water. In some cases the injury may be so mild that it may not be recalled by the patient [7]. Infection via an oral route is suspected in patients with diarrhea as the only clinical manifestation. Unusual routes of exposure include scuba diving or near drowning [8] and following surgical procedures [9,10].

Classical manifestations usually begin with localized cellulites and adenitis at the site of the trauma or wound, with rapid progression to overwhelming septicemia and the development of

multiple visceral abscesses. The common sites of abscess formation are the liver, lung and spleen. Rare brain abscesses have been reported. Other presentations include pneumonia [11], diarrhea, urinary tract infections, conjunctivitis, purulent otitis externa with mastoiditis [12], meningitis [13] and osteomyelitis. Deaths resulting from *C. violaceum* infections are generally caused by the fulminant septicemia. Most of the reported patients are young. In a series from the United States, 11 out of 12 patients had a mean age of 15 years [14]. In India, out of the 8 reported cases, 7 occurred in children less than 10 years [3].

There have been reports that *C. violaceum* septicemia occurs more commonly in patients with chronic granulomatous disease, neutrophil dysfunction, and severe polymorphonuclear G6PD deficiency [15,16]. In these conditions, polymorphonuclear leucocytes and monocytes lack the ability to produce oxygen metabolites required to kill phagocytised bacteria [17], which renders the patient susceptible to develop septicemia and dissemination of infection to multiple organs. Moreover, virulent strains of *C. violaceum* can produce an endotoxin that withstands attack from phagocytic cells. The rapid progression of this infection and the high fatality rate prevents evaluation for these underlying immunodeficiencies in most cases. Hence anyone surviving a *C. violaceum* infection must be evaluated for these underlying conditions [12]. However, there is no association with other immunodeficiency states such as HIV infection, diabetes mellitus, cancer chemotherapy, immunosuppressant drugs or long-term steroid use [18].

Eight cases of *C. violaceum* infection have been reported in India so far (Table 1) [3,13,19-25]. Our case, however, is the first case of septicemic *C. violaceum* infection in an adult reported from southern India. Seven out of the eight cases reported from India were children. The only adult patient reported from India was a young male from the Eastern part of the country who had a non-resolving wound abscess on his right leg without septicemia [23].

Our patient did not have any history of trauma or wounds, so, the route of infection remains unknown. He was known to have systemic lupus erythematoses and had been on long-term intermittent steroids. There were no other obvious immunodeficiencies. There is no literature on whether steroid usage predisposes to *C. violaceum* infection. Septic arthritis is an unusual manifestation of this illness and probably occurred

secondary to bacteremia, though there were no visceral abscesses on ultrasonography.

There are no clinical trials evaluating different treatments. However, antibiotics that have been successfully used are co-trimoxazole and flouroquinolones such as ciprofloxacin. Other antibiotics to which *C. violaceum* is usually susceptible *in vitro* are chloramphenicol, tetracycline and carbapenems. They are usually resistant to penicillins and narrow-spectrum cephalosporins. Susceptibility to aminoglycosides and broad-spectrum third-generation cephalosporins is variable [25].

This case report highlights that though *C. violaceum* is rare, it can be a cause of community-acquired septicaemia, especially among patients in a rural community with a history of contact with soil and stagnant water, in newborns, and in patients presenting with a melioidosis-like syndrome. Since it is a potentially fatal infection, a high degree of clinical suspicion and appropriate therapy at the earliest stages is absolutely essential.

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